

**Gushue v Levy**

2012 NY Slip Op 32483(U)

September 27, 2012

Supreme Court, New York County

Docket Number: 106645/05

Judge: Jeffrey K. Oing

Republished from New York State Unified Court System's E-Courts Service.  
Search E-Courts (<http://www.nycourts.gov/ecourts>) for any additional information on this case.

This opinion is uncorrected and not selected for official publication.

SUPREME COURT OF THE STATE OF NEW YORK — NEW YORK COUNTY

PRESENT: **JEFFREY K. OING**  
J.S.C.

PART 48

Index Number : 106645/2005  
**GUSHUE, KATHLEEN**  
vs.  
**LEVY, NORMAN**  
SEQUENCE NUMBER : 002  
SUMMARY JUDGMENT

INDEX NO. \_\_\_\_\_  
MOTION DATE \_\_\_\_\_  
MOTION SEQ. NO. \_\_\_\_\_  
MOTION CAL. NO. \_\_\_\_\_

on this motion to/for \_\_\_\_\_

PAPERS NUMBERED

Notice of Motion/ Order to Show Cause — Affidavits — Exhibits ...

Answering Affidavits — Exhibits \_\_\_\_\_

Replying Affidavits \_\_\_\_\_

\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

Cross-Motion:  Yes  No

Upon the foregoing papers, It is ordered that this motion

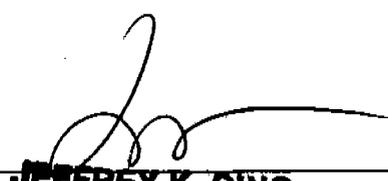
"This motion is decided in accordance with the annexed decision and order of the Court."

**FILED**

SEP 28 2012

NEW YORK  
COUNTY CLERK'S OFFICE

Dated: 9/27/12

  
**JEFFREY K. OING** J.S.C.

Check one:  FINAL DISPOSITION  NON-FINAL DISPOSITION

Check if appropriate:  DO NOT POST  REFERENCE

SUBMIT ORDER/ JUDG.

SETTLE ORDER/ JUDG.

MOTION/CASE IS RESPECTFULLY REFERRED TO JUSTICE FOR THE FOLLOWING REASON(S):

SUPREME COURT OF THE STATE OF NEW YORK  
COUNTY OF NEW YORK: IAS PART 48

-----x

KATHLEEN GUSHUE,

Plaintiff,

Index No.: 106645/05

-against-

Mtn Seq. No.: 002

ESTATE OF NORMAN LEVY, NFL REGAL, LLC  
D/B/A THE REGAL COMPANY, ALICE HUGHES,  
individually, CHARLES DEIDAN,  
individually, DEIDAN INDUSTRIES,  
DEIDAN INDUSTRIES, LTD, AND EJN  
CORPORATION,

DECISION AND ORDER

Defendants.

**FILED**

-----x

JEFFREY K. OING, J.:

SEP. 28 2012

Parti **NEW YORK  
COUNTY CLERKS OFFICE**

Plaintiff Kathleen Gushue. Defendants Estate of Norman  
Levy, NFL Regal, LLC d/b/a The Regal Company, and Alice Hughes  
(collectively referred to as the "Regal defendants") and  
defendant EJM Corp. d/b/a Tribeca Potters ("EJM") (the Regal  
defendants and EJM collectively referred to as "defendants").

**Factual Background**

From 1994 to 2004, plaintiff and EJM were tenants in a  
Manhattan building located at 443/451 Greenwich Street (the  
"premises"), which the Regal defendants owned. EJM rented space  
on the second floor for a pottery studio. Plaintiff, an artist  
and art administrator, rented studio space on the third and  
fourth floor. In 1996, plaintiff partitioned the third floor  
space and subleased it to other artists. During plaintiff's  
tenancy on the third floor, she complained of odors emanating  
from EJM's second floor space. In its ten year tenancy, EJM

received three violations from the NYC Department of Environmental Protection ("DEP") - one violation for failing to have the necessary operating certificate and two violations for unspecified odors.

In spring 2003, plaintiff observed a resting tremor and decreased agility in her right hand. In summer 2003, Anthony Santiago, MD examined plaintiff. Thereafter, a number of doctors evaluated plaintiff to treat her condition and to determine possible causes of her condition. One of plaintiff's experts, Paul A. Nausieda, MD, a board certified neurologist specializing in movement disorders, explained the neurological conditions as follows:

Parkinsonism is a term describing those neurological disorders which have clinical features including a tremor at rest (as opposed to tremor during voluntary movement), rigidity of the muscles, slowness of movement, and alterations in gait and station that result in a flexed posture and slowed shuffling gait with a tendency to fall rearward. Idiopathic Parkinson Disease is the most commonly encountered form of Parkinsonism and is a disorder of unknown etiology primarily affecting individuals over the age of 60. Parkinsonism is also seen in a variety neurodegenerative conditions ... in which pathologic changes occur in areas not affected by Idiopathic Parkinson Disease. ... Secondary Parkinsonism is a term used to describe syndromes with a known etiology and includes various forms of drug induced Parkinsonism that are caused by pharmacologic agents. ... Other forms of Parkinsonism are caused by various neurotoxins which damage areas of the brain that regulate body movement and posture. ... Manganese Poisoning results in pathologic damage to the Globus Pallidus and the Substantia Nigra.

(Nausieda Affirm., ¶ 12[d]).

Defendants proffer the affidavits of Cheryl Waters, MD, FRCP; Karl D. Kieburtz, MD, MPH; and Jose A. Obeso, MD in support of their respective summary judgment motions. Dr. Waters, a neurologist, examined plaintiff in October 2004 and indicated that plaintiff, "suffered from idiopathic Parkinson's Disease for which she was adequately medicated with levodopa at the time of [] examination" (Waters Aff., ¶ 3).

Dr. Kieburtz, a neurologist, specializing in the diagnosis and treatment of movement and inherited neurological disorders, and familiar with "published literature and studies concerning whether exposure to certain environmental toxicants, such as heavy metals (including manganese) and pesticides, might be a cause of Parkinson's disease" stated, "it is my opinion, to a reasonable degree of medical certainty, (and undisputed based upon the records provided to me) that Plaintiff suffers from idiopathic Parkinson's disease" which is "unrelated to any kiln fumes, including any manganese in the kiln fumes, to which she may have been exposed in the past" (Kieburtz Aff., ¶¶ 7, 9, 10).

Dr. Obeso opined, "without hesitation, based on my own scientific work, my regular attendance at scientific and medical meetings, and my position as Co-Editor-in-Chief of the major journal in the field, that it is not generally accepted among neurologists or movement disorder specialists that manganese causes Parkinson's disease" (Obeso Aff., ¶ 14).

In opposition, plaintiff proffers the affidavits of Elan D. Louis, MD; Paul A. Nausieda, MD; Edward A. Olmsted, CIH, CSP

("Olmsted"); Monona Rossol, MS, MFA ("Rossol"); and Stephen King, PhD, MPH.

Dr. Louis, a professor of neurology and a board certified neurologist, is currently plaintiff's treating neurologist. When he examined plaintiff in February and April 2005, he described plaintiff's clinical features as follows:

rest tremor, rigidity, bradykinesia and gait and balance changes (flexed posture, reduced arm swing, imbalance), which are four cardinal feature of Parkinson's disease. These clinical neurological features are found in patients with Idiopathic Parkinson's Disease..., Gene-Induced Parkinson's Disease..., and manganese-Induced Parkinson's disease (i.e. Parkinson's disease whose cause is Manganese toxicity).

(Louis Affirm., ¶ 7[a]).

Dr. Louis concluded:

It is highly likely that [plaintiff] was exposed to manganese containing fumes at her work place. She reports having worked for a prolonged period of time over a kiln with faulty exhaust, she complained of smelling fumes on many occasions to which she reports having had an allergic reaction, and her workplace ambient manganese levels were measured and were elevated. Manganese is a know (sic) neurotoxin that has been associated in epidemiological studies with Parkinson's disease and Parkinsonism. Given a known exposure to Parkinson's disease etiological agent (prior to her development of her clinical syndrome), the fact that her clinical features, PET results and MRI result are consistent with Manganese-induced Parkinson's disease, and the fact that Idiopathic Parkinson's disease (a rare disease to begin with) is even more rare in young women, her Parkinson's disease, to a reasonable degree of medical certainty, is Manganese induced Parkinson's disease rather than Parkinson's disease with no identified cause (Idiopathic Parkinson's disease).

(Louis Affirm., ¶ 7[e]).

Dr. Nausieda examined plaintiff in February 2010 and determined "within a reasonable degree of medical probability that [plaintiff] suffers from Manganese-Induced Parkinson's disease" (Nausieda Affirm., ¶ 6). Dr. Nausieda based his conclusion on plaintiff's symptoms since 2003, his observation of plaintiff's resting tremor on the right side and prominent antigavity tremor of the right hand, and "her symptoms [being] resistant to levodopa reversal though she has some responsiveness to levodopa replacement" (Nausieda Affirm., ¶ 12[h]). Dr. Nausieda also relied on the "[f]orensic testing of [plaintiff's] work space [which] demonstrated the presence of abnormally high levels of manganese (270 ug/ft<sup>2</sup>), as well as a number of other metals" (Nausieda Affirm., ¶ 12[i]).

The forensic testing referred to by Dr. Nausieda was conducted by Olmsted, an industrial hygienist and certified safety professional, retained by plaintiff. Olmsted conducted surveys, primarily on the third floor of the premises, on December 12, 2002, August 5, 2004, and May 25, 2005. Olmsted noted that the "[r]eadings for Lead, Manganese, Zinc, Iron, Copper, Chromium, Cobalt, Silver and Cadmium were elevated" (Olmsted Aff., ¶ 7). Olmsted also noted:

Although there are no standards for settled manganese, there are government limits set for lead, which are 40 micrograms per square foot. In one complaint areas (sic), levels of lead were 300 times the limit for surface dust. Manganese levels were 10 times the amount in the complaint area than in the control area.

(Olmsted Aff., ¶ 7).

Olmsted concluded, "within a reasonable degree of professional certainty, that the kilns were improperly designed and operated, as was the exhaust ventilation ... [and] caused kiln exhausts containing metals including manganese to enter the third floor studio space resulting in exposure to the occupants including [plaintiff]" (Olmsted Aff., ¶ 7).

Plaintiff also offered the affidavit of Rossol, a chemist and industrial hygienist, who opined that as a result of an insufficient kiln ventilation system plaintiff was exposed to metal fumes during daily firings, including excessive levels of manganese in her studio (Rossol Aff., ¶¶ 3, 4, 5). Rossol based her conclusions on general knowledge about clay and glazes, particularly EJM's use of Jasper; internet postings of defendant Emily Pearlman; knowledge of EnviroVent, the vent used by EJM, and its ventilation system and; the Olmsted Report (Rossol Aff., ¶¶ 7, 8, 9, 11, 13, 15, 18, 20).

#### **The Instant Action**

Plaintiff Kathleen Gushue commenced this action against the Regal defendants and EJM alleging that she was exposed to noxious toxic fumes emanating from EJM's second floor studio. Specifically, in her bill of particulars, plaintiff alleges exposure to toxic fumes from "metals including lead, cadmium, manganese, copper, zinc, nickel, iron, chromium and cobalt." She alleges in her bill of particulars the following:

12. Set forth a detailed statement of the injuries allegedly sustained by plaintiff and a description of those claimed to be permanent.

Ans: The plaintiff was diagnosed with Parkinson's disease on or about July 15, 2003. Parkinson's Disease is a neurological syndrome usually resulting from a deficiency of the neurotransmitter dopamine as the consequence of degenerative, vascular, or inflammatory changes in the basal ganglia; characterized by rhythmic muscular tremors, rigidity of movement, festination, droopy posture, and masklike faces. Plaintiff has tremor and stiffness in her right hand. She is right hand dominant. Tremor occurs at rest. Action tremor is slightly more prominent on the right than on the left side of her body, with loss of amplitude and early fatigability when doing repetitive tasks.

(Moving Papers, Ex. C, ¶ 12).

The Regal defendants move, pursuant to CPLR 3212, for summary judgment dismissing the complaint and all cross-claims against them. EJM cross-moves for summary judgment dismissing the complaint and all cross-claims against it.

Defendants argue that plaintiff's claim that she suffers from manganese-induced Parkinson's disease is an impossibility. Therefore, because no causal connection can be proven, the complaint must be dismissed. Plaintiff contends that the critical determination is whether the injury to her nervous system is idiopathic or caused by exposure to manganese. Thus, succinctly stated, the issue is whether manganese or manganese as contained in kiln fumes caused plaintiff's Parkinson's disease. I heard extensive arguments on August 19, 2011.

#### **Discussion**

The principle is well accepted that in a toxic tort action plaintiff must establish: (1) exposure to a particular toxin; (2) general causation - the toxin plaintiff was exposed to is capable

of causing the illness or condition she allegedly suffers; and (3) specific causation - plaintiff was exposed to sufficient levels of the toxin to cause the illness or condition she allegedly suffers (Parker v. Mobil Oil Corp., 7 NY3d 434, 446 [2006]).

Defendants challenge plaintiff's ability to satisfy the three prongs set forth in Parker, particularly because plaintiff pleads Parkinson's disease, not Parkinsonism or manganese-induced Parkinsonism. Plaintiff asserts, however, that her experts' opinions demonstrate that she was exposed to manganese or manganese as contained in kiln fumes, that the manganese was capable of causing her injury and that the level of manganese to which she was exposed was sufficient to cause her injury.

**(1) Plaintiff's exposure to manganese**

Defendants argue that plaintiff cannot establish that she was exposed to manganese or manganese as contained in kiln fumes. To demonstrate exposure to manganese, plaintiff relies on (1) the two DEP violations for unspecified odor; (2) the Olmsted Report; and (3) the expert and doctor opinions.

First, plaintiff's reliance on the DEP violations is misplaced because the violations do not specify that the complained of odor originated from manganese or manganese as contained in the kiln fumes emanating from EJM's second floor.

Second, defendants challenge plaintiff's reliance on the Olmsted Report. Defendants assert that the Olmsted Report is unreliable because (1) it did not provide any measurements or

test results for airborne levels of manganese or manganese as contained in kiln fumes in plaintiff's third floor studio or EJM's second floor space for any period of time; (2) it did not show plaintiff's exposure to manganese; (3) it did not demonstrate the source, date or any measured change of manganese surface concentration; and (4) there is no measurement of manganese as contained in the compounds used by EJM (Sapon Affirm., ¶ 8). More specifically, the Olmsted 2002 Indoor Air Quality Survey did not test for manganese and the 2004 Metals Testing Surveys took an air sample and swipe samples. The June 2004 survey used a test swipe from the Paula Elliott Studio, a self-contained room located above the kiln room on the third floor and the August 2004 survey used a test swipe taken from the A/J studio, where plaintiff worked. The June 2004 survey indicated "elevated" levels of manganese relative only to the control sample taken in the third floor kitchen. The results of the August 2004 survey did not reveal elevated levels of manganese. Additionally, the surveys were conducted within plaintiff's space, but neither survey occurred in EJM's second floor studio space. While the Olmsted Report confirmed manganese as one of several metals identified in plaintiff's work space, there is no recognized level at which manganese exposure is known to be toxic (Olmsted Aff., ¶ 7). Thus, at best, the Olmsted Report revealed that manganese was present and indicated a 10% increase in manganese levels between the test and the control.

Notwithstanding defendants' challenge to the consistency and reliability of the Olmsted Report, the record, which includes the competing physician and expert opinions, is clear - plaintiff was exposed to manganese. The next question is whether defendants have demonstrated an absence of a factual issue with respect to general and specific causation as a consequence of plaintiff's exposure.

**(2) Whether manganese is capable of causing manganese-induced Parkinson's disease**

Defendants argue that general causation cannot be established because the medical and scientific communities do not generally accept the premise that exposure to manganese as contained in kiln fumes causes Parkinson's disease.

When a claimant is alleging toxic cause, an expert must "reliably rule out reasonable alternative causes of [the alleged harm] or idiopathic causes" (Barbaro v Eastman Kodak Company, 26 Misc 3d 1124(A) [Sup Ct, Nassau County 2010]).

Here, plaintiff proffers the opinion of Rossol, who primarily relied on the Olmsted Report, which indicated elevated levels of lead, also a recognized neurotoxin, in the "complaint room" at 200 ug/ft<sup>2</sup> and in the control "kitchen" at 5100 ug/ft<sup>2</sup> (Rossol Aff., ¶ 30). Rossol acknowledged the high levels of lead, but did not offer any explanation as to why plaintiff suffered from manganese exposure and not lead exposure, merely stating, "[w]hile the issue here is not lead exposure to [plaintiff], but rather that the presence of lead in relation to

the smaller amounts of other metals support the conclusion that manganese and all the other metals found were released by kilns" (Rossol Aff., ¶ 33). Also, Rossol did not consider prior levels of manganese in plaintiff's work space or the supplies used by other artists on the third floor.

Additionally, Dr. King, familiar with medical, scientific and toxicological studies related to the "absorption, distribution, biotransformation (metabolism), and excretion of manganese compounds through inhalation and oral ingestion, as well as the mechanisms of manganese-induced neurotoxicity, and its ability to cause deleterious effects and damage to the brain and to the central nervous system" opined "based on a reasonable degree of toxicological and scientific certainty, the development of Plaintiff, Kathleen Gushue's signs and symptoms of Parkinsonism are directly related to her exposure to manganese in fumes that were released from kilns located on the second floor in the building that she occupied on the third floor" (King Aff., ¶¶ 3, 7 [emphasis added]). Although Dr. King referenced a litany of studies and literature in an effort to bolster his conclusion that plaintiff's exposure is related to manganese poisoning or manganese-induced Parkinsonism, he failed to link manganese to plaintiff's claim of manganese-induced Parkinson's disease.

Dr. Louis noted, "although there is literature stating that patients with manganese-induced parkinsonism do not have asymmetry, rest tremor, and response to levodopa, this literature is erroneous and in fact, there are many reports of patients with

manganese-induced Parkinson's disease who have the features either alone or in combination" (Louis Affirm., Ex. 1). Dr. Louis failed, however, to identify the erring literature and does not offer cases that share similarities to plaintiff's condition.

Dr. Nausieda applied differential diagnosis to determine that plaintiff's condition was manganese-induced Parkinson's disease, ruling out idiopathic Parkinson's disease (see Cornell v 360 West 51<sup>st</sup> Street Realty LLC, et al., 95 AD3d 50, 61 [1st Dept 2012]) (differential diagnosis as an accepted scientific procedure so long as the agent considered to cause the injury is in fact capable of causing the alleged injury). Dr. Nausieda's medical and scientific opinion, however, is belied by plaintiff's alleged condition. Indeed, Dr. Nausieda conceded that there are "no reported cases of manganism caused by pottery kilns ... in the medical literature," but still concluded based on the presence of manganese in pottery products and plaintiff's alleged exposure to manganese that plaintiff suffered from manganese-induced Parkinsonism (Nausieda Aff., ¶¶ 10, 12[k] [emphasis added]). While Dr. Nausieda cited literature finding a relationship between manganese exposure and Parkinsonism, the dispute before me stems from plaintiff's alleged exposure to excessive levels of manganese, resulting in manganese-induced Parkinson's disease, which is distinguishable from idiopathic Parkinson's disease. Despite similar symptoms, "manganese induced neurological disease" damages the globus pallidus and idiopathic Parkinson's

disease damages the pars compacta of the substantia nigra

(Nausieda Affirm., ¶ 14).

Dr. Nausieda stated:

Clinical descriptions of manganese poisoning generally include Parkinsonian examination features but may include unique features such as abnormal postures (dystonias), and prominent psychiatric symptoms (manganese madness). In other cases the examination may mimic idiopathic Parkinson Disease to a degree which makes differentiation difficult. The presence of an action or antigravity tremor may suggest a diagnosis of manganese toxicity, as may the early appearance of behavioral changes and cognitive symptoms, but in many cases the occupational history and the age of onset may be the most important differentiating features.

(Nausieda Affirm., ¶ 16).

While Dr. Nausieda declared that "Manganese poisoning needs to be considered as a cause of Parkinsonism in patients with a recognized exposure history and any of the atypical features" he failed to conclude that plaintiff is in fact suffering from manganese-induced Parkinsonism or Manganism, but rather diagnoses plaintiff's condition as manganese-induced Parkinson's disease, a condition unsupported by his own affidavit or the record.

Plaintiff also attempts to analogize the facts herein with a case where a male factory worker responsible for dumping fifty pound bags of manganese dioxide into a mixture five to six times a day in addition to sweeping his work area. The attempt is unavailing. The factory worker worked in two different ceramic factories for over thirteen years, both with poor ventilation systems. After being treated for manganese intoxication, the factory worker returned to work at the ceramic factory and

remained asymptomatic. In contrast, after ENJ relocated in 2004, plaintiff continued to be symptomatic.

Furthermore, plaintiff's reliance on pending Multi-District Litigation, In Re Welding Fumes Products Liability Litigation 2005 WL 1868046 (ND Ohio, J. O'Malley), to demonstrate that there is scientific or medical literature corroborating that fumes from pottery kilns contain numerous toxins, including manganese, and inhalation of manganese dust causes damage to the central nervous system exhibited by Parkinson's disease symptoms, is misplaced. In In Re Welding Fumes, the court found insufficient epidemiological evidence linking welding fumes to Parkinson's disease, but, nevertheless, identified enough "reliable" and "other known evidence" to support the assertion that exposure to welding fumes can cause, contribute to, or accelerate a Parkinsonian syndrome which doctors will diagnose as Parkinson's disease (Id.). Furthermore, the Multi-District Litigation concerned manganese as it related to welding fumes and not manganese as contained in kiln fumes.

Plaintiff argues that the absence of studies linking manganese as contained in kiln fumes to Parkinson's disease is not fatal to her claim because "the fact that there was no textual authority directly on point to support the expert's opinion is relevant only to the weight to be given the testimony but does not preclude its admissibility" (Zito v Zabarsky, 28 AD3d 42 [2d Dept 2006]). Plaintiff maintains that her diagnosis

is less relevant than the crucial question of what caused plaintiff to experience her symptoms.

To challenge plaintiff, defendants point to their experts, specifically, Dr. Kieburtz who declared that "[n]o study has ever demonstrated a causal link between manganese exposure and Parkinson's disease, and I am not aware of a study exploring a potential association between manganese in kiln fumes and Parkinson's disease" (Kieburtz Aff., ¶ 21). Dr. Kieburtz noted that there were studies that explored the association between manganese and Parkinson's disease, that none of the studies studied kiln fumes, and that there is no statistically significant association between exposure to manganese and the onset of Parkinson's disease (Kieburtz Aff., ¶ 22). Notably, Dr. Kieburtz acknowledged, "I am aware that it has been reported in the medical and scientific literature that exposure to manganese may cause a neurological syndrome as 'manganism' or 'manganese-induced parkinsonism'" (Kieburtz Aff., ¶ 33). Dr. Kieburtz, however, distinguished between 'manganism' and 'parkinsonism' as distinctly different from manganese-induced Parkinson's disease, which is plaintiff's alleged condition (Kieburtz Aff., ¶ 35).

Dr. Obeso also stated that "[a]lthough many hundreds of environmental factors have been proposed as potential causes of Parkinson's disease, to date no cause has been established or accepted for this progressive, neurodegenerative disease" (Obeso Aff., ¶ 12).

Here, plaintiff has failed to raise a factual issue as to whether there is a nexus between manganese or manganese as contained in kiln fumes and Parkinson's disease (Fraser v Townhouse Corp. et al., 57 AD3d 416 [1st Dept 2008]) (where plaintiff's expert evidence fell short because none of the medical literature supported their opinions that plaintiff's exposure caused his condition). In Fraser v 301-52 Townhouse Corp., 57 AD3d 416 [1st Dept 2008], the Appellate Division dismissed a complaint even though plaintiff established an association between symptoms and mold based on the rationale that plaintiff's expert "conflat[ed] the distinct concepts of association and causation" (Fraser at 418-419).

Accordingly, the record lacks medical or scientific evidence that exposure to manganese as contained in kiln fumes causes manganese-induced Parkinson's disease. Indeed, absent from the record is any demonstration that peer reviewed articles or studies exist to suggest otherwise.

**(3) The amount of manganese plaintiff was exposed is known to cause manganese-induced Parkinson's disease**

Assuming arguendo that plaintiff were able to raise a factual issue with respect to general causation, plaintiff must still satisfy Parker's third prong - specific causation. To satisfy the third prong, plaintiff must demonstrate that the level of manganese exposure caused her Parkinson's disease. Defendants argue that plaintiff cannot establish the necessary threshold dosage of concentration of manganese in pottery kiln

fumes that causes Parkinson's disease. Defendants further assert that even if plaintiff were to demonstrate the threshold level of exposure, plaintiff cannot show that she was exposed to manganese at levels that exceeded the threshold.

Plaintiff maintains that the Olmsted Report demonstrated that due to insufficient ventilation she was exposed to manganese in excessive amounts which accumulated from the EJM kilns located on the second floor below plaintiff's studio.

In Parker, the Court of Appeals held that so long as the methods used by an expert to establish causation are generally accepted in the scientific community quantifying exposure levels or providing dose-response relationships is not necessary if the expert is able to demonstrate sufficiently that plaintiff's exposure caused her condition (Parker, 7 NY3d at 446). In that regard, the Court upheld the Appellate Division's finding that despite citing studies demonstrating a link between benzene and AML, plaintiff's experts "did not prove the causal connection between the exposure to benzene in gasoline" (Id. at 446). As a result, the Court reasoned that even if plaintiff's experts had established that he was exposed to the threshold level of benzene they could not show his exposure to exceed the threshold level and, therefore, any conclusion would be speculative (Id.).

Here, based on the Olmsted Report, Dr. Nausieda concluded that plaintiff's exposure to manganese in her work space was "abnormally high." Yet, Dr. Nausieda neither offers an accepted threshold level at which manganese exposure is toxic, nor does he

identify a study to compare plaintiff's case. Thus, without further substantiation the notion that plaintiff's exposure to manganese as contained in kiln fumes caused her manganese-induced Parkinson's disease is conclusory.

Although Dr. Louis concluded that, "it is highly likely that [plaintiff] was exposed to manganese containing fumes at her work place," he acknowledged in an April 7, 2005 letter that plaintiff's MRI was "unremarkable," her PET scan was normal and her blood manganese levels did not indicate any levels of toxicity (Louis Affirm., ¶ 7[e], Ex. 1). Indeed, absent from the record is any evidence suggesting either how much exposure to manganese will render plaintiff susceptible to toxic poisoning, or, alternatively, the extent of plaintiff's exposure to manganese (Coratti v. The Wella Corp., 56 AD3d 343, 344 [1st Dept 2008]). Quite simply, given plaintiff's failure to provide quantifiable measures to show her exposure met or surpassed the necessary threshold, plaintiff has failed to raise a factual issue with regard to specific causation.

#### **Conclusion**

At oral argument, plaintiff's counsel stated:

[It] [j]ust befuddles me that if the term Parkinsonism had been used, it's an entirely different case. I mean, you know, it's a question of the constellation of symptoms. And they know what the issues are and they know what the injury is and they know what the exposure is.

(Transcript, August 19, 2011, p. 34).

Counsel's statement is an oversimplification of the pleading requirements and issues in this action. In so much that individuals suffering from manganism or manganese-induced Parkinsonism may exhibit features similar to those common to idiopathic Parkinson's disease, given that the record fails to demonstrate that plaintiff's diagnosis was manganism or manganese-induced Parkinsonism, or that any expert testimony established that manganese or manganese as contained in kiln fumes causes Parkinson's disease, plaintiff has failed to raise an issue of fact with respect to causation, general or specific.

At oral argument, plaintiff's attorney inquired:

So I (sic) this case going to come down to in the BP the termed it manganese-induced Parkinson's disease (sic) and they said it can't be, well if Parkinson's disease include the symptoms of Parkinsonism, then where is the harm? We have alleged the exposure, being manganese. We have alleged the injury. If we had mistakenly called it beriberi disease, does that mean that they're not aware of the claim?

(Tr., at pp. 19-20).

Contrary to counsel's statement, individuals afflicted with Parkinsonism may not have Parkinson's disease. Thus, allegations must be precise because idiopathic Parkinson's disease, manganese-induced Parkinson's disease, Parkinsonism or manganese-induced Parkinsonism are not mutually exclusive. Thus, plaintiff could allege Parkinsonism and suffer from Parkinson's disease, but to assert that she suffered from manganese-induced Parkinson's disease and also call her condition manganese-induced Parkinsonism is improper. The record is clear that manganism or

Index No. 106645/05  
Mtn Seq. No. 002

Page 20 of 20

manganese-induced Parkinsonism and idiopathic Parkinson's disease are distinguishable because they have different causes, impact different parts of the brain, and have different features.

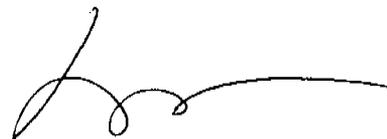
Accordingly, the Regal defendants' motion and defendant EJM's cross-motion for summary judgment dismissing the complaint are granted, and the action is hereby dismissed.

ORDERED that the Regal defendants' motion and defendant EJM's cross-motion for summary judgment are granted, and the complaint is dismissed; and it is further

ORDERED that the Clerk is directed to enter judgment accordingly.

This memorandum opinion constitutes the decision and order of the Court.

Dated: 9/27/12



HON. JEFFREY K. OING, J.S.C.

**FILED**

SEP 28 2012

NEW YORK  
COUNTY CLERK'S OFFICE